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THE HEART IN EMPHYSEMA*

Pulmonary emphysema is becoming a more frequent problem, which probably is related to the increased number of older persons in the poplation. There are two important aspects of cardiac involvement to be considered in emphysema. First, the development of cor pulmonale indicates that the patient has reached an advanced stage in the disease and has a poor prognosis. Secondly, emphysema and cor pulmonale are frequently incorrectly diagnosed. As a result the patient may receive improper therapy with disastrous results.

Pathologic Physiology

Some understanding of the pathologic physiology of pulmonary emphysema is essential to a discussion of the cardiac complications. The essential defect in emphysema is alteration in the elastic properties of the lungs associated with bronchiolar obstruction. Accompanying this condition are changes in the lung compartments and a diminution in the maximum rate of expiratory air flow. The residual volume of the lung is regularly increased, the vital capacity is reduced, and the total lung capacity is normal or increased. The impairment of the rate of expiratory air flow results in a decreased maximum breathing capacity and a decrease in the ratio of one second to total vital capacity. These alterations in ventilatory function are intimately related to the sensation of dyspnea which is the major clinical manifestation of the disease.

Patients with a mild to moderate degree of emphysema may exhibit no change in the blood gases. Hypoxia and hypercapnia commonly develop in the later stage of the disease. The decrease in the oxygen saturation and increase in the carbon dioxide tension of the arterial blood are related to uneven alveolar ventilation and unequal perfusion of alveoli with blood. Some alveoli are poorly ventilated and others well ventilated. This can be demonstrated by the abnormal way gases mix with air in the lungs. There is also evidence that some alveoli are inadequately supplied with blood, resulting in impaired exchange of gas in these alveoli. Increase in carbon dioxide tension in the blood is compensated by a decrease in the chloride and an increase in the buffer base and bicarbonate content of the blood. As a result the blood pH remains normal, or perhaps is slightly decreased.

Cor pulmonale is invariably associated with pulmonary hypertension. Elevation in pulmonary arterial pressure is in large part responsible for the hypertrophy of the right ventricle and the development of right ventricular failure in pulmonary emphysema. Pulmonary arterial hypertension is a result of an increase in resistance in the small arteries or arterioles; the pulmonary capillary pressure remains normal. The increase in pulmonary arterial pressure is highly correlated with the decrease in oxygen saturation of the arterial blood. This correlation is of such a degree that one can predict with some accuracy the pulmonary arterial pressure from knowledge of the oxygen saturation of the hemoglobin of the arterial blood. It seems likely that, by some mechanism as yet unknown, hypoxia increases pulmonary arterial resistance. Structural alterations in the vascular bed as a result of pulmonary disease also undoubtedly contribute to the increased vascular resistance.

There has been some controversy regarding the cardiac output in patients with emphysema. Early studies suggested that the cardiac output was increased in most of these patients. More recent studies describe the cardiac output as normal or slightly decreased. Patients with emphysema and *heart failure* have a higher cardiac output than patients with heart failure on the basis of hypertension or valvular disease. It is significant that an increase in blood volume has been found in patients with pulmonary emphysema and cor pulmonale. The increase is chiefly in the red cell volume. In some instances a large increase in hematocrit reading may occur and contribute to the pulmonary hypertension by increasing the viscosity of the blood.

It is important to remember that the primary disease in emphysema is pulmonary, the abnormal hemodynamics being due to the structural changes in the lungs and to hypoxia. On the other hand, the changes in the lung in congestive heart failure secondary to hypertension or valvular disease are related to alterations in the pulmonary circulation. The characteristic change in the lung in the latter is a decrease in the compliance of the lung which is related to the increase in pulmonary vascular pressure, increase in pulmonary blood

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volume and pulmonary edema. As a result, the vital capacity and total lung capacity are decreased, whereas the residual volume is normal. Alveolar ventilation is adequate and the carbon dioxide tension is normal or slightly reduced. Hypoxia may be present but is usually not severe unless pulmonary edema is marked.

Clinical Picture

The usual history of the patient with emphysema and heart failure is one of progressive dyspnea and cough of several years' duration. Recent exacerbation of these symptoms, often secondary to an acute respiratory infection, together with edema of the legs, are often presenting complaints. The patient is dyspneic at rest and is commonly most comfortable in the sitting position. Cyanosis is present and may be severe. The neck veins are often distended when the patient is in the sitting position. Cardiac enlargement usually cannot be detected because of the hyperresonance of the chest. The liver is enlarged and edema is present. Signs of hydrothorax may be found.

The determination of the presence of right heart failure in a patient with severe emphysema may not be simple. Patients with emphysema may develop edema of the legs in the absence of right heart failure, particularly if the legs remain in a dependent position for long periods of time. The liver is displaced downward by the low diaphragm and therefore can be readily felt. The best criterion of right heart failure is elevation of right ventricular diastolic pressure, which is reflected by an elevation in systemic venous pressure. Roentgenologic signs of enlargement of the right chambers of the heart are useful evidence of failure. Because of the frequent vertical position of the heart, such a change may be difficult to evaluate in a plain PA-roentgenogram. Serial films may demonstrate significant alteration in heart size, and the electrocardiogram may reveal evidence of right ventricular hypertrophy.

Differential Diagnosis

Of paramount importance is the differentiation of pulmonary emphysema with heart failure from congestive heart failure of other etiology with associated pulmonary congestion and edema. It has been mentioned that the patient with emphysema will have a long history of dyspnea and cough. On physical examination it may be helpful to ascertain the depth of cyanosis. Characteristically, patients with emphysema and heart failure are deeply cyanotic, which has therefore given rise to the term "black cardiac."

Caution must be exercised in the interpretation of the chest examination. While many patients with emphysema exhibit an increase in anteroposterior diameter of the chest, limited expansion and obliteration of the cardiac and hepatic dullness, these same findings may be present in elderly patients without the physiologic abnormalities of pulmonary emphysema. Of some aid is the fact that the level of the diaphragm tends to be lower in the patients with emphysema than in those with pulmonary congestion and edema. Although the heart in emphysema with heart failure is enlarged, physical examination will not give good evidence of the increase in its size. For this reason, a forceful apex impulse to the left of the midclavicular line almost excludes the diagnosis of cor pulmonale. Significant murmurs are usually absent, and auricular fibrillation is uncommon in cor pulmonale.

As mentioned, the electrocardiogram and roentgenogram of the chest may give evidence of right heart enlargement in emphysema. High-peaked P waves may be seen in the electrocardiogram. It should be remembered that the roentgenographic picture of the lungs in pulmonary emphysema may not be diagnostic. The diaphragm is usually low in emphysema, whereas elevation of the diaphragm is common in pulmonary congestion and edema. The circulation time may be of some help in that it is shorter in patients with emphysema than in patients with heart failure related to hypertension or valvular disease.

Vital capacity is reduced in the patient with pulmonary emphysema or with pulmonary congestion and edema, but in emphysema the residual volume is increased. This determination is helpful in diagnosis but requires special equipment. The maximum rate of expiratory air flow is markedly diminished in emphysema. This is reflected by a marked diminution in maximum breathing capacity and one-second vital capacity. In the patient with pulmonary congestion and edema the rate of expiratory air flow is not markedly impaired unless pulmonary edema is severe.

Determination of oxygen saturation, carbon dioxide content and pH of arterial blood, is of great aid if facilities for these determinations are available. In emphysema with heart failure the arterial oxygen saturation is reduced to a considerable extent, the CO_2 tension is elevated, and the pH is decreased to a variable degree. In contrast, in pulmonary congestion and edema the CO_2 tension is variable from normal to low, and the pH is normal or high. The decrease in O_2 saturation is variable but often less than in emphysema. The CO_2 content may be high following mercurial diuresis.

Treatment

In the treatment of cor pulmonale complicating pulmonary emphysema, the major effort should be directed toward improving pulmonary ventilation and restoring the oxygen saturation and carbon dioxide tension of the arterial blood toward normal. Right heart failure in pulmonary emphysema is most commonly precipitated by a respiratory infection such as bronchitis. Treatment of the bronchitis with antibiotics may reduce bronchial obstruction and improve ventilation of the functioning alveoli. This in turn may lead to an increase in oxygen saturation of the arterial blood, a decrease in the pressure in the pulmonary artery and disappearance of the signs of right heart failure. Penicillin administered intramuscularly in large amounts appears to be the most

effective means of treating the infection. Bronchodilator drugs are also helpful. These can be given conveniently by means of a nebulizer and either isuprel or epinephrine may be effective. The technique of administration is important and has been described by Miller. Morphine, demerol and sedatives should be avoided, as their use may result in a decrease in alveolar ventilation.

Hypoxia in patients with severe emphysema appears to be responsible, at least in part, for the cardiac complications, and if they become severe may lead to death. Hence, the use of oxygen in patients with severe emphysema and hypoxia appears logical. Unfortunately, the administration of oxygen may be accompanied by depression of ventilation with increasing stupor, and finally coma. These changes are associated with a rise in the carbon dioxide tension and a fall in the pH of the arterial blood. This decrease in ventilation is related to the removal of the hypoxic stimulus to breathing, which has become the major driving force for respiration. Carbon dioxide becomes a relatively ineffective stimulus to breathing in the patient with severe emphysema. Because of this complication, oxygen must be used with great caution. If oxygen is administered, the patient should be carefully observed to determine whether his level of consciousness improves. If he becomes more stuporous, the use of oxygen should be stopped or, if it needs to be continued, mechanical assistance to breathing should be instituted. Either an intermittent positive pressure device, such as the Bennett apparatus or a tank respirator, may be used. The patient must be carefully observed. He is often confused and irrational and will fight the respirator, and for this reason the therapy may be ineffective in improving ventilation.

Removal of secretions from the respiratory tract is important. If the patient is stuporous he loses his cough reflex and secretions rapidly accumulate in the respiratory tract. These should be removed by suction. When accumulation of secretions in the trachea and upper bronchi constitutes a major problem, bronchoscopy may be necessary, or tracheostomy to permit adequate suction and to decrease the bronchial dead space.

The treatment of the failing myocardium is of much less importance than the relief of bronchial obstruction and hypoxia. Digitalis has been demonstrated to increase the cardiac output and decrease right ventricular diastolic pressure. For this reason it would appear to be indicated in those patients with evident right ventricular failure. A low-salt diet and diuretics may be useful in combating edema. Diamox has been recommended, not only because of its diuretic effect, but also because it may tend to increase ventilation of the lungs. The latter effect is the result of production of a metabolic acidosis. However, the drug does not appear to have great practical value for long-term therapy.

Summary

Heart failure complicating pulmonary emphysema is becoming an increasingly frequent problem. It is important to recognize this clinical entity because the treatment differs from that of heart failure of other etiology. Right ventricular failure in emphysema is related to the development of pulmonary hypertension. This in turn is correlated with the severity of hypoxia.

Treatment of heart failure complicating emphysema should be directed toward relieving bronchial obstruction and improving alveolar ventilation. Antibiotic therapy is important because bronchial infection is usually present. Sedatives must be avoided and oxygen therapy administered with caution.

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REFERENCES

1. Ebert, R. V.: Pulmonary emphysema. Ann. Rev. Med. 7: 123, 1956.
2. Harvey, R. M., Ferrer, M. I., and Cournand, A.: Treatment of chronic cor pulmonale. Circulation 7: 932, 1953.
3. Harvey, R. M., Ferrer, M. I., Richards, D. W., Jr., and Cournand, A.: Influence of chronic pulmonary disease on heart and circulation. Am. J. Med. 10: 719, 1951.
4. Miller, W. F.: Consideration of improved methods of nebulization therapy. New England J. Med. 251: 589, 1954.
5. Segal, M. S., and Dulfano, M. J.: Chronic Pulmonary Emphysema: Physiopathology and Treatment. Modern Medical Monographs, 8. New York, Grune and Stratton, Inc., 1953.

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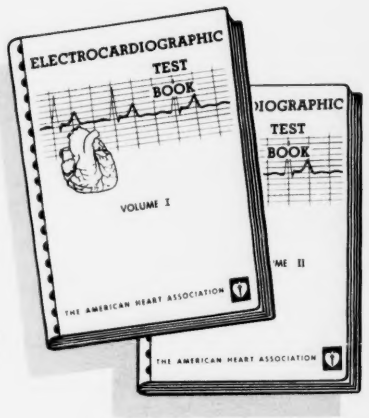

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